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Time: 10:15-12:15
Room: Room 1.40

Carriage and laboratory detection of multi-drug resistant bacteria in low and middle-income countries

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Multidrug resistance is now emerging worldwide at an alarming rate among Gram negatives, causing both community-acquired and nosocomial infection. The most important emerging resistance traits are those reported*Enterobacteriaceae* since they represent the vast majority of human infections. The emerging resistance traits in *Enterobacteriaceae* are dominated by the clavulanic acid-inhibited ß-lactamases (ESBLs) and the carbapenemases which hydrolyze most if not all ß-lactams including the carbapenems. Detection of infected patients rely on classical identification of infecting agent and susceptibility testing. Two biochemical tests, the ESBL NDP and the Carba NP tests, have been recently developed for the early detection of ESBL- or carbapenemase -associated resistance traits. Those tests are rapid, sensitive, specific and cost-effective. Their implementation can be done worldwide in any clinical microbiology laboratory. Their extended usage may significantly improve the management and outcome of infected patients.

Detection of carriers is important mostly for patients hospitalized in ICU and important surgery. Taking in account the prevalence rate of the ESBL and carbapenemase producers and the dynamic of hospital-based outbreaks, detection of carriers is important for detecting mostly *K.pneumoniae*, E*nterobacter* sp, . (and not *E. coli*) which produce an ESBL and any enterobacterial species which produce a carbapenemase. This detection is based on a screening using a selective plate containing a cephalosporin (ESBL producer) or a carbapenem (carbaepenemase producer). A series of commercialized and home-made (SUPERCARBA culture medium) are readily available. Strains growing on those media shall be screened rapidly for ESBL producer b yusing the ESBL NDP test and for carbapenemase producer (Carba NP test). This strategy shall prevent the development of outbreaks in hospitals among severely-ill patients and preserve as much as possible the antibiotics of last resort.

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Antibiotic resistance and virulence

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Antimicrobial resistance, either by mutation or acquisition of resistance determinants harboured by mobile genetic elements, may confer a biological cost for the bacteria. This fitness cost can affect the growth rate *in vitro* or the survival in the host or in the environment or the virulence capacity. However, bacteria can evolve and adapt to reduce this cost, by compensatory mutations or fine regulation of resistance expression.Antibiotic resistance may also be associated with increased virulence or transmission and may play in role in global spread and dominance of certain resistant bacteria, pending the plasmid type. In this regard, a successful international sequence type, CTX-M producing *E. coli* ST131, was shown to have a competitive advantage over other isolates of *E. coli*, promoting its clonal expansion and dominance over less virulent and/or more susceptible *E. coli* clones. In addition, Pitout and colleagues demonstrated that NDM-1-producing *E. coli* ST131 had significantly more virulence factors than NDM-1-producing *E. coli* ST101. In contrast, amongst carbapenemase-producing *K.pneumoniae*, the same authors found no significant *in vitro* differences in the presence of virulence factors between ST11, ST147, ST258, and the other sequence types of carbapenemase-producing *K.pneumoniae* globally, whilst Lavigne and colleagues showed in their *in vivo* model, an increase of virulence of KPC-2 producing strains belonging to ST-258. In fact, regarding the various CPE, Nordmann and colleagues had also recently demonstrated discordance amongst such pathogens, and a possible “fitness cost” *in vivo* due to the fact that not all carbapenemases are expressed to the same extent of their *in vitro* expression. This has potential therapeutic implications for carbapenems and other antibiotic therapy particularly for NDM and OXA-48-like infections but not apparently for KPC infections according to their results. In summary, although recent studies have demonstrated that antibiotic resistance generally confers a reduction in fitness e.g. MDR *P.aeruginosa* and penicillin resistant *N.meningitidis*; the opposite may also be true e.g. CA MRSA, *agr* dysfunction amongst bacteraemic MRSA and fluoroquinolone resistance of the NAP1/027 *C.difficile* epidemic strains, and therefore appears to be distinct for every specie and resistant genotype, pending compensatory evolution.

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